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APPLICATION NO.	FILING DATE	FIRST NAMED INVENTOR	ATTORNEY DOCKET NO.	CONFIRMATION NO.
10/791,166	03/01/2004	Israel R. Charo	02307K-085041US	3893

20350 7590 02/23/2005

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EXAMINER	
NICHOLS, CHRISTOPHER J	
ART UNIT	**PAPER NUMBER**
1647	

DATE MAILED: 02/23/2005

Please find below and/or attached an Office communication concerning this application or proceeding.

Office Action Summary	Application No.	Applicant(s)	
	10/791,166	CHARO ET AL.	
	Examiner	Art Unit	
	Christopher J Nichols, Ph.D.	1647	

-- The MAILING DATE of this communication appears on the cover sheet with the correspondence address --

Period for Reply

A SHORTENED STATUTORY PERIOD FOR REPLY IS SET TO EXPIRE 3 MONTH(S) FROM THE MAILING DATE OF THIS COMMUNICATION.

- Extensions of time may be available under the provisions of 37 CFR 1.136(a). In no event, however, may a reply be timely filed after SIX (6) MONTHS from the mailing date of this communication.
- If the period for reply specified above is less than thirty (30) days, a reply within the statutory minimum of thirty (30) days will be considered timely.
- If NO period for reply is specified above, the maximum statutory period will apply and will expire SIX (6) MONTHS from the mailing date of this communication.
- Failure to reply within the set or extended period for reply will, by statute, cause the application to become ABANDONED (35 U.S.C. § 133). Any reply received by the Office later than three months after the mailing date of this communication, even if timely filed, may reduce any earned patent term adjustment. See 37 CFR 1.704(b).

Status

1)☒ Responsive to communication(s) filed on *01 March 2004*.

2a)☐ This action is **FINAL**. 2b)☒ This action is non-final.

3)☐ Since this application is in condition for allowance except for formal matters, prosecution as to the merits is closed in accordance with the practice under *Ex parte Quayle*, 1935 C.D. 11, 453 O.G. 213.

Disposition of Claims

4)☒ Claim(s) *1-17* is/are pending in the application.

4a) Of the above claim(s) _____ is/are withdrawn from consideration.

5)☐ Claim(s) _____ is/are allowed.

6)☒ Claim(s) *1-17* is/are rejected.

7)☐ Claim(s) _____ is/are objected to.

8)☐ Claim(s) _____ are subject to restriction and/or election requirement.

Application Papers

9)☒ The specification is objected to by the Examiner.

10)☒ The drawing(s) filed on *01 March 2004* is/are: a)☐ accepted or b)☒ objected to by the Examiner.

Applicant may not request that any objection to the drawing(s) be held in abeyance. See 37 CFR 1.85(a).

Replacement drawing sheet(s) including the correction is required if the drawing(s) is objected to. See 37 CFR 1.121(d).

11)☐ The oath or declaration is objected to by the Examiner. Note the attached Office Action or form PTO-152.

Priority under 35 U.S.C. § 119

12)☐ Acknowledgment is made of a claim for foreign priority under 35 U.S.C. § 119(a)-(d) or (f).

a)☐ All b)☐ Some * c)☐ None of:

1.☐ Certified copies of the priority documents have been received.

2.☐ Certified copies of the priority documents have been received in Application No. _____.

3.☐ Copies of the certified copies of the priority documents have been received in this National Stage application from the International Bureau (PCT Rule 17.2(a)).

* See the attached detailed Office action for a list of the certified copies not received.

Attachment(s)

1) ☒ Notice of References Cited (PTO-892)
2) ☐ Notice of Draftsperson's Patent Drawing Review (PTO-948)
3) ☒ Information Disclosure Statement(s) (PTO-1449 or PTO/SB/08) Paper No(s)/Mail Date *6.1.04*.
4) ☐ Interview Summary (PTO-413) Paper No(s)/Mail Date. _____.
5) ☐ Notice of Informal Patent Application (PTO-152)
6) ☐ Other: _____.

DETAILED ACTION

Status of Application, Amendments, and/or Claims

1. The Preliminary Amendment filed 1 March 2004 has been received and entered in full.

Information Disclosure Statement

2. The information disclosure statement (IDS) filed 1 June 2004 fails to comply with 37 CFR 1.98(a)(1), which requires the following: (1) a list of all patents, publications, applications, or other information submitted for consideration by the Office; (2) U.S. patents and U.S. patent application publications listed in a section separately from citations of other documents; (3) the application number of the application in which the information disclosure statement is being submitted on each page of the list; (4) a column that provides a blank space next to each document to be considered, for the examiner's initials; and (5) a heading that clearly indicates that the list is an information disclosure statement. The information disclosure statement filed 1 June 2004 only contains sheets 3-5 of 5 sheets as noted in the IDS. The information in sheets 3-5 of the IDS have been considered. Applicant is invited to provide copies of sheets 1-2 with the response to this Office Action to complete the IDS.

Drawings

3. The drawings are objected to because Figures 1, 2, 3, 4, and 7 contain multiple subcomponents (i.e. "1A", "1B") which are not described in the Specification. A proposed drawing correction or corrected drawings are required in reply to the Office action to avoid abandonment of the application. The objection to the drawings will not be held in abeyance.

Specification

4. The disclosure is objected to because of the following informalities: typos "MoI", "Nat'l.", and "nrocesses" (**[0006]**); typo "Enzymolo^{g}y" (**[0015]**); typo "FIG. 1 llIllustrates" (**[0030]**); typo "■ -lactomase" (**[0066]**); typo "Ni"" (**[0130]**). Appropriate correction is required.

Claim Rejections - 35 USC § 112

The following is a quotation of the first paragraph of 35 U.S.C. 112:

> The specification shall contain a written description of the invention, and of the manner and process of making and using it, in such full, clear, concise, and exact terms as to enable any person skilled in the art to which it pertains, or with which it is most nearly connected, to make and use the same and shall set forth the best mode contemplated by the inventor of carrying out his invention.

5. Claims **1-9** are rejected under 35 U.S.C. 112, first paragraph, because the specification, while being enabling for *a method of inhibiting a condition characterized by monocytic infiltrates, wherein said method comprises*

> *administering to a patient a therapeutically effective amount of an MCP-1 antagonist in a suitable pharmaceutical carrier, wherein said MCP-1 receptor antagonist binds to an MCP-1 receptor polypeptide and wherein said antagonist is an antibody or binding fragment thereof which binds to a MCP-1 receptor,*

does not reasonably provide enablement for *practicing said method using other antibodies with no specified target, other agents*. The specification does not enable any person skilled in the art to which it pertains, or with which it is most nearly connected, to make the invention commensurate in scope with these claims.

6. The invention is drawn to a therapeutic method comprising administering an antagonist of MCP-1 receptor.

7. The Specification teaches that MCP-1 receptor also known as monocyte chemoattractant protein-1 receptor, CCR2, CKR-2, MCP-1RA, MCP-1RB, and CC-chemokine receptor 2 [see US 6312689 (LaRosa) 6 November 20001 (**IDS**)] is the receptor for MCP-1 (monocyte chemoattractant protein-1), a chemokine from the C-X-C subfamily. MCP-1 and consequently MCP-1R are involved in conditions characterized by monocytic infiltrates such as rheumatoid arthritis, atherosclerosis, and alveolitis. In addition, MCP-1 plays a key role in the recruitment of monocyte-macrophages into developing atherosclerotic lesions. MCP-1 can also induce tumoricidal activity. The Examiner notes that the Specification provides an enabling disclosure for the use of antibodies which bind the MCP-1 receptor but not the full range of antagonists claimed.

8. The specification fails to provide any guidance for the successful treatment of any conditions characterized by monocytic infiltrates comprising administering a therapeutically effective amount of an MCP-1 antagonist. However, sufficient guidance is present for use of an antibody or antibody binding fragment which binds the MCP-1 receptor. As for any other agents, the resolution of the various complications in regards to evaluating the antagonistic activity of as of yet characterized antagonists is highly unpredictable. As such, one of skill in the art would have been unable to practice the invention without engaging in undue trial and error experimentation. In order to practice the invention using the specification and the state of the art as outlined below, the quantity of experimentation required to practice the invention as claimed would require the *de novo* determination of formulations of MCP-1 receptor antagonists as well

as signs and symptoms of conditions characterized by monocytic infiltrates to correlate with therapeutic regiments. In the absence of any guidance from the specification, the amount of experimentation would be undue and one would have been unable to practice the invention over the scope claimed.

9. Additionally, a person skilled in the art would recognize that predicting the efficacy of using MCP-1 antagonists which must first be identified by a screening method based solely on prophetic suggestion as highly problematic (see MPEP §2164.02). Thus, although the specification prophetically considers and discloses general methodologies of using the claimed therapeutic method, such a disclosure would not be considered enabling since the state of therapy for MCP-1 related conditions is highly unpredictable and complex. The factors listed below have been considered in the analysis of enablement [see MPEP §2164.01(a) and *In re Wands*, 858 F.2d 731, 737, 8 USPQ2d 1400, 1404 (Fed. Cir. 1988)]:

(A) The breadth of the claims;
(B) The nature of the invention;
(C) The state of the prior art;
(D) The level of one of ordinary skill;
(E) The level of predictability in the art;
(F) The amount of direction provided by the inventor;
(G) The existence of working examples; and
(H) The quantity of experimentation needed to make or use the invention based on the content of the disclosure.

10. The following references are cited herein to illustrate the state of the art of monocytic infiltrate conditions and therapies thereof.

11. On the breadth of the claims, the art recognizes that an "antagonist" can pertain to chemical entities, pharmaceutical compositions, proteins, peptides, non-peptide compounds, animal tissue extracts, nucleic acids, antisense molecules, peptidomimetic, transformed cells,

antibodies, antibody fragments, cyclic peptides, inhibitors, vegetable extracts, cell extracts, synthetic agents, biologically derived substances as well as proteinaceous substances, known, and unknown compounds. The instant claims present an invitation to experiment, first to identify antagonists, then characterize their properties, and then determine therapeutic regiments for their use.

12. On the nature of the invention, Sheikine & Hansson (2004) "Chemokines and atherosclerosis." Annals of Medicine **36**(2): 98-118 teach that atherosclerosis is a complex disease of chronic inflammation of a vessel wall involving lesions, lipid build up (plaques), possible infection, and a wide array of immunological signaling molecules and cells (pp. 98-99). MCP-1 attracts cells to the lesion while other chemokines affect atherogenesis by a variety of other mechanisms such as regulation of cell proliferation, angiogenesis, and scavenging (Table 1). Thus the skilled artisan is confronted by a complex array of intertwining immunological agents, any of which may interact with or be affected by a MCP-1 receptor antagonist requiring undue experimentation in the absence of concrete guidance as to the structure and function of the MCP-1 antagonist.

13. On the state of the prior art, Iyonaga *et al.* (May 1994) "Monocyte Chemoattractant Protein-1 in Idiopathic Pulmonary Fibrosis and Other Interstitial Lung Disease." Human Pathology **25**(5): 455-463 teaches that MCP-1 is involved in idiopathic pulmonary fibrosis but the mechanism of its action and its role in the pathology was unknown at the time (pp. 455). In addition, Tang *et al.* (May 1994) "Cytokine Expression, Upregulation of Intracellular Adhesion Molecule-1, and Leukocyte Infiltration in Experimental Tubulointersitital Nephritis." Laboratory Investigation **70**(5): 631-638 teaches that MCP-1 expression is increased in an experimental

model of nephritis (Figure 1 & Table 1). Also Sousa *et al.* (February 1994) "Increased Expression of the Monocyte Chemoattractant Protien-1 in Bronchial Tissue from Asthmatic Subjects." Am. J. Respir. Cell Mol. Biol.**10**(2): 142-147 teaches that MCP-1 is involve in bronchial asthma (Figures 1-3). Thus the prior art teaches that that MCP-1 is involved in idiopathic pulmonary fibrosis, nephritis, and bronchial asthma but is silent as to the identity of MCP-1 antagonists, their activities, and their usefulness in treating idiopathic pulmonary fibrosis, nephritis, and/or bronchial asthma. This would require *de novo* experimentation to first identify antagonists, then characterize them, and the test the so identified antagonists for therapeutic value.

14. On the level of predictability in the art, MacDermott (December 1996) "Alterations of the mucosal immune system in inflammatory bowel disease." J Gastroenterol. **31**(6): 907-16 teaches that the normal intestinal immune system is a balance of pro-inflammatory and anti-inflammatory cells and molecules which are carefully regulated to promote a healthy mucosa without damage to the host tissue. If this delicate regulatory balance is disturbed, nonspecific stimulation and activation can lead to increased amounts of potent destructive immunological and inflammatory molecules. For instance, lipopolysaccride (LPS) from resident microflora is capable of activating macrophages and T lymphocytes to release potent inflammatory cytokines, including interleukin-1 (IL-1), interleukin-6 (IL-6), and tumor necrosis factor alpha (TNF-α). IL-1 and TNF-α stimulate epithelial cells, endothelial cells, macrophages, and fibroblasts to secrete potent chemotactic cytokines, such as monocyte chemoattractant protein-1 (MCP-1) which recruits macrophages and granulocytes into the inflamed mucosa (pp. 907, 911-912). Thus the intestinal immune system and mucosa are sensitive to perturbations of the cytokine balance. This

presents a high level of unpredictability if one were to add antagonists of MCP-1 which may trigger this inflammatory response and thus have no useful therapeutic effect as instantly claimed.

15. Furthermore on the predictability in the art, Gong & Clark-Lewis (February 1995) "Antagonists of Monocyte Chemoattractant Protein 1 Identified by Modification of Functionally Critical NH2-terminal Residues." J. Exp. Med. **181**(2): 631-640 teach that most truncated forms of MCP-1 lack specific binding or any MCP-1 activity (pp. 631). For instance, a single amino acid deletion, a 2-76 residue fragment, showed 300-fold lower activity than the 76 residue full-length MCP-1 (Figure 4). In addition, other analogues including the 7-76, 8-76, 9-76, 10-76, and 11-76 lacked any detectable activity (Figures 4-5). Thus the skilled artisan is confronted with an unpredictable art as minor modifications in the native ligand obliterate binding and activity. As such the skilled artisan is confronted with an unpredictable milieu in which to achieve the goal of the full scope of the claims as instantly presented.

16. In order to practice the invention using the specification and the state of the prior art as outlined above, the quantity of experimentation required to practice the invention as claimed would require the identification, cloning, and characterization of MCP-1 receptor antagonists that possess the desired properties. In the absence of any guidance from the specification, the amount of experimentation would be undue and one would have been unable to practice the invention over the scope claimed.

17. Claims **10-17** are rejected under 35 U.S.C. 112, first paragraph, because the specification, while being enabling for *a method of inhibiting MCP-1 receptor polypeptide comprising*

administering a therapeutically effective amount of an MCP-1 antagonist in a suitable pharmaceutical carrier wherein said antagonist is an antibody or binding fragment thereof which binds to a MCP-1 receptor,

does not reasonably provide enablement for *practicing said method using other antibodies with no specified target, other agents*. The specification does not enable any person skilled in the art to which it pertains, or with which it is most nearly connected, to make the invention commensurate in scope with these claims.

18. The invention is drawn to a therapeutic method comprising administering an antagonist of MCP-1 receptor.

19. The Specification teaches that MCP-1 receptor also known as monocyte chemoattractant protein-1 receptor, CCR2, CKR-2, MCP-1RA, MCP-1RB, and CC-chemokine receptor 2 [see US 6312689 (LaRosa) 6 November 20001 (**IDS**)] is the receptor for MCP-1 (monocyte chemoattractant protein-1), a chemokine from the C-X-C subfamily. MCP-1 and consequently MCP-1R are involved in conditions characterized by monocytic infiltrates such as rheumatoid arthritis, atherosclerosis, and alveolitis. In addition, MCP-1 plays a key role in the recruitment of monocyte-macrophages into developing atherosclerotic lesions. MCP-1 can also induce tumoricidal activity. The Examiner notes that the Specification provides an enabling disclosure for the use of antibodies which bind the MCP-1 receptor but not the full range of antagonists claimed.

20. The specification fails to provide any guidance for the successful inhibition of MCP-1 receptor comprising administering a therapeutically effective amount of an MCP-1 antagonist. However, sufficient guidance is present for use of an antibody or antibody binding fragment

which binds the MCP-1 receptor. As for any other agents, the resolution of the various complications in regards to evaluating the antagonistic activity of as of yet characterized antagonists is highly unpredictable. As such, one of skill in the art would have been unable to practice the invention without engaging in undue trial and error experimentation. In order to practice the invention using the specification and the state of the art as outlined below, the quantity of experimentation required to practice the invention as claimed would require the *de novo* determination of formulations of MCP-1 receptor antagonists to correlate with MCP-1 receptor inhibition. In the absence of any guidance from the specification, the amount of experimentation would be undue and one would have been unable to practice the invention over the scope claimed.

21. Additionally, a person skilled in the art would recognize that predicting the efficacy of using MCP-1 antagonists which must first be identified by a screening method based solely on prophetic suggestion as highly problematic (see MPEP §2164.02). Thus, although the specification prophetically considers and discloses general methodologies of using the claimed method which encompasses therapies, such a disclosure would not be considered enabling since the state of MCP-1 antagonists is highly unpredictable and complex. The factors listed below have been considered in the analysis of enablement [see MPEP §2164.01(a) and *In re Wands*, 858 F.2d 731, 737, 8 USPQ2d 1400, 1404 (Fed. Cir. 1988)]:

(A) The breadth of the claims;
(B) The nature of the invention;
(C) The state of the prior art;
(D) The level of one of ordinary skill;
(E) The level of predictability in the art;
(F) The amount of direction provided by the inventor;
(G) The existence of working examples; and

(H) The quantity of experimentation needed to make or use the invention based on the content of the disclosure.

22. The following references are cited herein to illustrate the state of the art of monocytic infiltrate conditions and therapies thereof.

23. On the breadth of the claims, the art recognizes that an "antagonist" can pertain to chemical entities, pharmaceutical compositions, proteins, peptides, non-peptide compounds, animal tissue extracts, nucleic acids, antisense molecules, peptidomimetic, transformed cells, antibodies, antibody fragments, cyclic peptides, inhibitors, vegetable extracts, cell extracts, synthetic agents, biologically derived substances as well as proteinaceous substances, known, and unknown compounds. The instant claims present an invitation to experiment, first to identify antagonists, then characterize their properties, and then determine therapeutic regiments for their use.

24. On the nature of the invention, Sheikine & Hansson (2004) "Chemokines and atherosclerosis." Annals of Medicine **36**(2): 98-118 teach that atherosclerosis is a complex disease of chronic inflammation of a vessel wall involving lesions, lipid build up (plaques), possible infection, and a wide array of immunological signaling molecules and cells (pp. 98-99). MCP-1 attracts cells to the lesion while other chemokines affect atherogenesis by a variety of other mechanisms such as regulation of cell proliferation, angiogenesis, and scavenging activity (Table 1). Thus the skilled artisan is confronted by a complex array of intertwining immunological agents, any of which may interact with or be affected by a MCP-1 receptor antagonist requiring undue experimentation in the absence of concrete guidance as to the structure and function of the MCP-1 antagonist.

25. On the state of the prior art, Iyonaga *et al.* (May 1994) "Monocyte Chemoattractant Protein-1 in Idiopathic Pulmonary Fibrosis and Other Interstitial Lung Disease." Human Pathology **25**(5): 455-463 teaches that MCP-1 is involved in idiopathic pulmonary fibrosis but the mechanism of its action and its role in the pathology was unknown at the time (pp. 455). In addition, Tang *et al.* (May 1994) "Cytokine Expression, Upregulation of Intracellular Adhesion Molecule-1, and Leukocyte Infiltration in Experimental Tubulointersititial Nephritis." Laboratory Investigation **70**(5): 631-638 teaches that MCP-1 expression is increased in an experimental model of nephritis (Figure 1 & Table 1). Also Sousa *et al.* (February 1994) "Increased Expression of the Monocyte Chemoattractant Protien-1 in Bronchial Tissue from Asthmatic Subjects." Am. J. Respir. Cell Mol. Biol.**10**(2): 142-147 teaches that MCP-1 is involve in bronchial asthma (Figures 1-3). These references are of interest as the claimed method inherently encompasses therapies. And as such even though the prior art teaches that that MCP-1 is involved in idiopathic pulmonary fibrosis, nephritis, and bronchial asthma it is silent as to the identity of MCP-1 antagonists, their activities, and their usefulness in treating idiopathic pulmonary fibrosis, nephritis, and/or bronchial asthma. This would require *de novo* experimentation to first identify antagonists, then characterize them, and the test the so identified antagonists for the desired inhibitory property.

26. On the predictability in the art, Gong & Clark-Lewis (February 1995) "Antagonists of Monocyte Chemoattractant Protein 1 Identified by Modification of Functionally Critical NH2-terminal Residues." J. Exp. Med. **181**(2): 631-640 teach that most truncated forms of MCP-1 lack specific binding or any MCP-1 activity (pp. 631). For instance, a single amino acid deletion, a 2-76 residue fragment, showed 300-fold lower activity than the 76 residue full-length MCP-1

(Figure 4). In addition, other analogues including the 7-76, 8-76, 9-76, 10-76, and 11-76 lacked any detectable activity (Figures 4-5). Thus the skilled artisan is confronted with an unpredictable art as minor modifications in the native ligand obliterate binding and activity. As such the skilled artisan is confronted with an unpredictable milieu in which to achieve the goal of the full scope of the claims as instantly presented.

27. In order to practice the invention using the specification and the state of the prior art as outlined above, the quantity of experimentation required to practice the invention as claimed would require the identification, cloning, and characterization of MCP-1 receptor antagonists that possess the desired property. In the absence of any guidance from the specification, the amount of experimentation would be undue and one would have been unable to practice the invention over the scope claimed.

28. Claims **1, 2, 3, 4, 9, 10, 11, 12,** and **13** are rejected under 35 U.S.C. 112, first paragraph, as containing subject matter which was not described in the specification in such a way as to reasonably convey to one skilled in the relevant art that the inventor(s), at the time the application was filed, had possession of the claimed invention.

29. The independent claims require an "MCP-1 receptor antagonist" while practicing the claimed methods but fail to elaborate on its structure thus implying that it is not known or must be confirmed. The art recognizes that "agent" can pertain to chemical entities, pharmaceutical compositions, proteins, peptides, non-peptide compounds, animal tissue extracts, nucleic acids, antisense molecules, peptidomimetic, transformed cells, antibodies, antibody fragments, cyclic

peptides, inhibitors, vegetable extracts, cell extracts, synthetic agents, biologically derived substances as well as proteinaceous substances, known, and unknown compounds.

30. To provide adequate written description and evidence of possession of a claimed genus, the specification must provide sufficient distinguishing identifying characteristics of the genus. The factors to be considered include disclosure of complete or partial structure, physical and/or chemical properties, functional characteristics, structure/function correlation, methods of making the claimed product, or any combination thereof. In this case, the only factor present in the claim that is sufficiently disclosed is a recitation of a desired antagonist of an MCP-1 receptor with therapeutic and/or inhibitory properties. The specification does not identify any particular portion of the structure that must be conserved, nor does it provide a disclosure of structure/function correlation. The distinguishing characteristics of the claimed genus are not described. Accordingly, the specification does not provide adequate written description of the claimed genus.

31. To satisfy the written-description requirement, the specification must describe every element of the claimed invention in sufficient detail so that one of ordinary skill in the art would recognize that the inventor possessed the claimed invention at the time of filing. *Vas-Cath*, 935 F.3d at 1563; see also *Lockwood v. American Airlines, Inc.*, 107 F.3d 1565, 1572 [41 USPQ2d 1961] (Fed. Cir. 1997) (patent specification must describe an invention and do so in sufficient detail that one skilled in the art can clearly conclude that "the inventor invented the claimed invention"); *In re Gosteli*, 872 F.2d 1008, 1012 [10 USPQ2d 1614] (Fed. Cir. 1989) ("the description must clearly allow persons of ordinary skill in the art to recognize that [the inventor] invented what is claimed"). Thus, an applicant complies with the written-description requirement

"by describing the invention, with all its claimed limitations, not that which makes it obvious," and by using "such descriptive means as words, structures, figures, diagrams, formulas, etc., that set forth the claimed invention." *Lockwood*, 107 F.3d at 1572.

32. The instant Applicant does not provide the structure and/or identity of any MCP-1 receptor antagonists except for antibodies which bind SEQ ID NO: 2 or SEQ ID NO: 4. Thus the instant situation is most analogous to *University of Rochester v. G.D. Searle & Co.*, 68 USPQ2d 1424 (DC WNY 2003) and *University of Rochester v. G.D. Searle & Co. et al.* CAFC [(03-1304) 13 February 2004]. In *University of Rochester v. G.D. Searle & Co.* a patent directed to method for inhibiting prostaglandin synthesis in human host using an unspecified compound, in order to relieve pain without side effect of stomach irritation, did not satisfy written description requirement of 35 U.S.C. §112, since the patent described the compound's desired function of reducing activity of the enzyme PGHS-2 without adversely affecting PGHS-1 enzyme activity, but did not identify said compound, since invention consists of performing "assays" to screen compounds in order to discover those with desired effect. The patent did not name even one compound that assays would identify as suitable for practice of the invention, or provide information such that one skilled in art could identify a suitable compound. And since specification did not indicate that compounds are available in public depository, the claimed treatment method cannot be practiced without the compound. Thus the inventors cannot be said to have "possessed" the claimed invention without knowing of a compound or method certain to produce said compound. Thus said patent constituted an invitation to experiment to first identify, then characterize, and then use a therapeutic a class of compound defined only by their desired properties. As in the instant Application where a method of identifying the desired antagonists is

provided (**[0083]-[0085]**) but the actual antagonists are not, save for antibodies which bind SEQ ID NO: 2 or SEQ ID NO: 4.

33. Therefore the full breadth of the claim fails to meet the written description provision of 35 U.S.C. §112, first paragraph. Applicant is reminded that *Vas-Cath* makes clear that the written description provision of 35 U.S.C. §112 is severable from its enablement provision.

Summary

34. No claims are allowed.

35. The following articles, patents, and published patent applications were found by the Examiner during the art search while not relied upon for the instant rejection(s) are considered pertinent to the instant application:

a. US 2004/0151721 A1 (O'Keefe & Ponath) 5 August 2004

b. US 6,395,497 B1 (LaRosa) 28 May 2002

c. US 6,406,865 B2 (LaRosa) 18 June 2002

d. US 6,448,021 (LaRosa) 10 September 2002

Conclusion

Any inquiry concerning this communication or earlier communications from the examiner should be directed to **Christopher James Nichols, Ph.D.** whose telephone number is **(571) 272-0889**. The examiner can normally be reached on Monday through Friday, 8:00 AM to 5:00 PM. If attempts to reach the examiner by telephone are unsuccessful, the examiner's supervisor, **Brenda Brumback** can be reached on **(571) 272-0961**.

The fax number for the organization where this application or proceeding is assigned is **703-872-9306**.

Information regarding the status of an application may be obtained from the Patent Application Information Retrieval (PAIR) system. Status information for published applications may be obtained from either Private PAIR or Public PAIR. Status information for unpublished applications is available through Private PAIR only. For more information about the PAIR system, see **http://pair-direct.uspto.gov**. Should you have questions on access to the Private PAIR system, contact the Electronic Business Center (EBC) at **866-217-9197** (toll-free).

CJN
February 17, 2005